Introduction
The term “cosmetic” is used synonymously with “make-up” in the general population. However, it has a much broader definition and includes personal care products, hair care, nail products, and sunscreens.

There is an impressive array of cosmetic products available on the market today, with an even greater number of individual ingredients. The number of new products continues to increase and the rates of adverse cutaneous reactions are expected to rise. Therefore, given the widespread use of cosmetics, it is important to monitor their side-effects. It is estimated that the average woman uses 12 personal care products daily, which comprise 168 unique ingredients. The average man uses six personal care products each day with 85 unique ingredients. Skin care products have been found to account for the majority of cases of allergic contact dermatitis (ACD) to cosmetics, followed by hair care and nail cosmetics. The most common responsible cosmetic allergens are fragrances and preservatives.

Epidemiology
The prevalence of cosmetic allergy is estimated at less than 1% in the general population. However, since most people do not seek medical consultation for mild adverse reactions, the actual rates are likely much higher. Pooled data of seven different studies involving 30,207 patients patch tested for suspected contact dermatitis revealed that 9.8% of positive reactions were due to cosmetic allergens.

A recent Danish study showed that the prevalence of ACD to cosmetic allergens has doubled between 1990 and 1998. The majority of patients affected with ACD to cosmetic products are women between the ages of 20 and 55.

In addition, a study of 794 patients showed that 34% of patients would have been missed if they were only tested with NACDG (North American Contact Dermatitis Group) screening series of 65 allergens. A European analysis of 5911 cosmetic-allergic patients found that one-third reacted only to a personal product and no other allergen. Thus, while the optimal number of allergens for patch testing to cosmetics is not firmly established, testing for additional allergens using a specialized cosmetic series and the patient’s own personal products would capture about 30% of additional patients that otherwise would have been missed.

Clinical Features of ACD
ACD may have acute and chronic forms. Acutely, it presents with pruritic papules, vesicles, and bullae. Chronic forms are more common and present with pruritic, scaly papules and plaques distributed in areas of most contact with the offending allergen. The distribution provides very useful clinical clues about the possible causative agent. Occasionally, ACD may produce autoeczematization resulting in a widespread or generalized cutaneous eruption. Allergens may also be transferred from other persons or even pets, resulting in “connubial” or “consort” dermatitis.

The main differential diagnostic possibilities for ACD are exacerbation of atopic dermatitis or irritant contact dermatitis, both of which are far more common than allergic contact reactions.

ABSTRACT
Cosmetics are an important cause of allergic contact dermatitis (ACD). Fragrances and preservatives are the two most clinically relevant allergens found in cosmetic products. Patch testing remains the gold standard for identification of causative allergens. Common cosmetic allergens are reviewed. Practical methods of allergen avoidance are also discussed.

Key words: allergic contact dermatitis, fragrance, preservative, skin care
Classes of Allergens Responsible for ACD

Cosmetic ingredients can be classified into several categories: fragrances, preservatives, antioxidants, vehicles, ultraviolet absorbers, humectants, emollients, emulsifiers, acrylates, hair dyes, nail polish components, and others.

Preservatives and fragrances are the most frequently detected culprits; therefore, this review will primarily deal with these two classes of allergens.

Preservatives

Preservatives were identified as the most common cosmetic contact allergens in several recent studies. They can be classified into three broad categories: antimicrobials, antioxidants, and ultraviolet light absorbers. The antimicrobial agents can be further divided into formaldehyde preservatives, formaldehyde-releasers, and non-formaldehyde-releasing preservatives. Formaldehyde-releasing preservatives (FRP) include quaternium-15, diazolidinyl urea, imidazolidinyl urea, 2-bromo-2-nitropropane-1,3-diol, and DMDM hydantoin.

Non-formaldehyde-releasing preservatives include parabens, methylchloroisothiazolinone-methylisothiazolinone (MCI-MI), methyldibromoglutaronitrile-phenoxethanol (MDBGN-PE), and iodopropynyl butylcarbamate. Individuals allergic to formaldehyde may also be allergic to any of the FRPs. Formaldehyde-sensitized individuals may experience a flare of ACD with a number of foods, including cod fish, caviar, coffee, shiitake mushrooms, smoked ham, maple syrup, and aspartame.

Table 1 lists the top 20 NACDG screening allergens associated with cosmetic source in females.

For comparison, Tables 2a and 2b list the top 10 allergens from the North American Contact Dermatitis Group (NACDG) and the Mayo Clinic Contact Dermatitis Group (MCCDG) identified in all patients presenting for patch testing. It is evident that many of the top allergens are from cosmetic sources.

Fragrances

There are over 3000 different fragrances used in cosmetics today. Not surprisingly, fragrances represent the second most common group of cosmetic allergens. Available tools to assess for fragrance allergy are fragrance mix I (FMI), fragrance mix II (FMII),

<table>
<thead>
<tr>
<th>Rank</th>
<th>Allergen</th>
<th>No. of allergic reactions</th>
<th>% Females with allergy to cosmetic source, N = 1582</th>
<th>% Reactions in females associated with cosmetic source, N = 2920</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Quaternium-15 2%</td>
<td>323</td>
<td>20.4</td>
<td>11.1</td>
</tr>
<tr>
<td>2/3</td>
<td>Myroxylon pereirae (balsam of Peru) 25%</td>
<td>302</td>
<td>19.1</td>
<td>10.3</td>
</tr>
<tr>
<td>2/3</td>
<td>Fragrance mix 8%</td>
<td>302</td>
<td>19.1</td>
<td>10.3</td>
</tr>
<tr>
<td>4</td>
<td>p-Phenylenediamine 1%</td>
<td>247</td>
<td>15.6</td>
<td>8.5</td>
</tr>
<tr>
<td>5</td>
<td>Methylidibromoglutaronitrile-phenoxethanol 2%</td>
<td>131</td>
<td>8.3</td>
<td>4.5</td>
</tr>
<tr>
<td>6</td>
<td>Formaldehyde 1%</td>
<td>108</td>
<td>6.8</td>
<td>3.7</td>
</tr>
<tr>
<td>7</td>
<td>Tosylamide formaldehyde resin 10%</td>
<td>97</td>
<td>6.1</td>
<td>3.3</td>
</tr>
<tr>
<td>8</td>
<td>Cocamidopropyl betaine 1%</td>
<td>84</td>
<td>5.3</td>
<td>2.9</td>
</tr>
<tr>
<td>9</td>
<td>Glyceryl thioglycolate 1%</td>
<td>83</td>
<td>5.3</td>
<td>2.8</td>
</tr>
<tr>
<td>10/11</td>
<td>Diazolidinyl urea 1%</td>
<td>79</td>
<td>5.0</td>
<td>2.7</td>
</tr>
<tr>
<td>10/11</td>
<td>Diazolidinyl urea 1%</td>
<td>79</td>
<td>5.0</td>
<td>2.7</td>
</tr>
<tr>
<td>12</td>
<td>DMDM hydantoin 1%</td>
<td>77</td>
<td>4.9</td>
<td>2.6</td>
</tr>
<tr>
<td>13</td>
<td>Lanolin alcohol 30%</td>
<td>71</td>
<td>4.5</td>
<td>2.4</td>
</tr>
<tr>
<td>14/15</td>
<td>Imidazolidinyl urea 2%</td>
<td>70</td>
<td>4.4</td>
<td>2.4</td>
</tr>
<tr>
<td>14/15</td>
<td>Methylchloroisothiazolinone/methylisothiazolinone 100 ppm*</td>
<td>70</td>
<td>4.4</td>
<td>2.4</td>
</tr>
<tr>
<td>16</td>
<td>Methyl methacrylate 2%</td>
<td>65</td>
<td>4.1</td>
<td>2.2</td>
</tr>
<tr>
<td>17</td>
<td>Amidoamine 0.1%</td>
<td>63</td>
<td>4.0</td>
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<tr>
<td>18</td>
<td>Propylene glycol 30%</td>
<td>61</td>
<td>3.9</td>
<td>2.1</td>
</tr>
<tr>
<td>19</td>
<td>DMDM hydantoin 1%</td>
<td>58</td>
<td>3.7</td>
<td>2.0</td>
</tr>
<tr>
<td>20</td>
<td>Imidazolidinyl urea 2%</td>
<td>51</td>
<td>3.2</td>
<td>1.8</td>
</tr>
</tbody>
</table>

Table 1: Top 20 NACDG screening allergens associated with cosmetics in females

* Not in petrolatum; all others are
10 Most Common Allergens NACDG

<table>
<thead>
<tr>
<th>Allergen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nickel sulfate</td>
</tr>
<tr>
<td>Neomycin</td>
</tr>
<tr>
<td>Balsam of Peru</td>
</tr>
<tr>
<td>Fragrance mix</td>
</tr>
<tr>
<td>Thimerosal</td>
</tr>
<tr>
<td>Gold sodium thiosulfate</td>
</tr>
<tr>
<td>Quaternium-15</td>
</tr>
<tr>
<td>Formaldehyde</td>
</tr>
<tr>
<td>Bacitracin</td>
</tr>
<tr>
<td>Cobalt chloride</td>
</tr>
</tbody>
</table>

Table 2a: Top 10 list of common contact allergens from NACDG

10 Most Common Allergens MCCDG

<table>
<thead>
<tr>
<th>Allergen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nickel sulfate</td>
</tr>
<tr>
<td>Balsam of Peru</td>
</tr>
<tr>
<td>Gold sodium thiosulfate</td>
</tr>
<tr>
<td>Neomycin</td>
</tr>
<tr>
<td>Fragrance mix</td>
</tr>
<tr>
<td>Thimerosal</td>
</tr>
<tr>
<td>Cobalt chloride</td>
</tr>
<tr>
<td>Formaldehyde</td>
</tr>
<tr>
<td>Benzalkonium chloride</td>
</tr>
<tr>
<td>Bacitracin</td>
</tr>
</tbody>
</table>

Table 2b: Top 10 list of common contact allergens from MCCDG

and balsam of Peru. The components of these screening allergens are listed below:

**Fragrance Mix I** (8.0% in petrolatum)
- Amyl cinnamic alcohol 1.0%
- Cinnamic alcohol 1.0%
- Eugenol 1.0%
- Cinnamic aldehyde 1.0%
- Hydroxycitronellal 1.0%
- Geraniol 1.0%
- Isoeugenol 1.0%
- Oak moss absolute 1.0%
- Sorbitan sesquioleate (emulsifier) 5.0%

**Fragrance Mix II** (14.0% in petrolatum)
- Hydroxyisohexyl 3-cyclohexene carboxaldehyde (2.5%)
- Citral 1.0%
- Farnesol 2.5%
- Coumarin 2.5%
- Citronellol 0.5%
- Hexyl cinnamal 5.0%

Many of the specific fragrance ingredients are protected by the Fair Packaging and Labeling Act as they are considered trade secrets. It is important to keep in mind that many products labeled as “unscented”, “hypoallergenic”, or even “fragrance-free” do, in fact, contain masking fragrances.

Balsam of Peru
Balsam of Peru (Myroxylon pereirae resin) is an aromatic fluid that comes from the bark of the tree Myroxylon balsamum, a tree native to El Salvador. It is a complex mixture of many ingredients, all of which have not yet been completely identified. Key ingredients including benzoyl cinnamate, benzoyl benzoate, benzoic acid, vanillin, and nerodiol can be found in the following three groups of products: fragrance in perfumes and toiletries, flavorings in foods and drinks, and medications. In the past, FMI and BOP were able to detect approximately 90% of fragrance allergies. However, with the increasing number of fragrances and botanicals in use today, their screening ability is now estimated to be around 60%. Thus, FMII and a number of botanical extracts are now part of the 2010 NACDG screening series that comprise 70 allergens. Often, additional cosmetic and botanical series are required to diagnose fragrance allergy. Patients with contact allergy to BOP may also react to a number of substances that are well known cross-reactants with BOP (Table 3). Thus, patients should be appropriately counseled to avoid these agents.

**Cross Reacting Agents**

- Balsam of Tolu
- Benzoin
- Benzyl acetate
- Benzyl alcohol
- Cinnamic alcohol/cinnamic aldehyde
- Cinnamon oil
- Clove oil
- Essential oils of orange peel
- Eugenol
- Propolis

Table 3: Cross-reactants with balsam of Peru

**Practical Considerations and Clinical Pearls**

1. Choose allergens carefully: based on history, occupation, hobbies, and distribution of dermatitis. Patch testing may need to be expanded beyond the NACDG screening series to include, for example, a cosmetic/botanical supplemental series. This series may be indicated in patients using a variety of make-up products or for those who use “all natural” botanical products. Testing to personal care products may lead to identification of additional relevant allergens, as well as facilitate discovery of new and emerging allergens, as new compounds are being introduced at an escalating pace.

2. Have a good working knowledge of common allergens and their sources: this is critical for choosing the correct allergens to test as well as for counseling patients on allergen avoidance.

3. Have access to available resources: an excellent review of the main concepts of ACD is found in Contact Allergy: Alternatives for the 2007 NACDG Standard Screening Tray. Allergen information sheets are available to the members of the American Contact Dermatitis Society (ACDS) and can be found at www.contactderm.org. Identification of allergen-free products can be accomplished by generating a customized
product list with the use of the Contact Allergen Management Program (CAMP) available to the members of the ACDS. Multiple allergens can be entered to generate a “shopping list” of products that are safe to use in a patient with allergic contact dermatitis to their cosmetics.

**Conclusion**

Allergic contact dermatitis to cosmetics is an important cause of ACD overall. The main causes of cosmetic allergy are fragrances and preservatives. It is rewarding for both the patient and the physician if the responsible agent can be identified and subsequently removed from the patent’s environment. Patient satisfaction and compliance will also improve if meaningful counseling is provided, including detailed information on safe to use personal care products.

**References**

**Alcohol and Skin Disorders: With a Focus on Psoriasis**

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2DermSurgery Associates, Houston, TX, USA
3Department of Dermatology, The University of Texas Medical School at Houston, Houston, TX USA

**ABSTRACT**

Alcohol is a serious cause of morbidity and mortality in our society and is implicated in multiple health conditions, including hepatic failure, neurological damage, hematological disorders, and nutritional deficiencies, to name a few. Although alcohol induced cutaneous abnormalities can also cause significant morbidity, they tend to be overshadowed by the other disease states associated with alcohol use. In addition to the cutaneous stigmata linked to chronic alcoholic liver disease, alcohol can directly cause or exacerbate several skin conditions. In particular, alcohol misuse is implicated in the development of psoriasis and discoid eczema, as well as confers increased susceptibility to skin and systemic infections. Alcohol misuse might also exacerbate rosacea, porphyria cutanea tarda, and post adolescent acne. Herein, we review the evidence concerning the influences of alcohol in skin conditions with a focus on psoriasis.

**Key words:** alcohol drinking, psoriasis, risk factors, skin disorders

**Physiology of Alcohol Induced Toxicity**

Alcohol induces a wide range of physiological derangements in the human body. Alcohol is cytotoxic to the liver, leading to alcoholic steatosis, hepatitis and, at later stages, cirrhosis with systemic sequelae. Alcohol is also toxic to the bone marrow, particularly the T cells, which in turn leads to attenuated immune function.2-4 The cardiovascular system may also be adversely affected by excess alcohol use. Specifically, high output cardiac failure, hypertension, and peripheral vasodilatation may be consequences of chronic and acute alcohol intake.2,5 Finally, alcohol misuse results in a myriad of nutritional deficiencies, including vitamin and trace element deficiencies secondary to interference with proper intestinal absorption and poor nutrition. All of these physiological conditions can contribute to the development of cutaneous manifestations associated with alcohol consumption.

**Alcohol and Skin Disorders**

Cutaneous abnormalities have been associated with alcoholism and are either caused indirectly through impaired functioning of other organ systems or directly from the toxic effects of alcohol on the skin.

**Skin Changes Indirectly Caused by Alcohol**

The majority of cutaneous manifestations associated with excess alcohol use are indirectly mediated through the impairment of various organ systems.

Hepatic dysfunction impairs estrogen and bile salt metabolism, resulting in characteristic findings of spider angiomata, palmar erythema, and pruritis.2,3,5 Male alcoholics are consequently hyperestrogenic.2 In addition to high estrogen levels, testosterone production is also inhibited, further exacerbating the problem. Direct inhibition of testosterone production leads to gynecomastia, which presents as a disappearance and redistribution of body and pubic hair and female pattern fat redistribution.2 Caput medusae and hemorrhoids are the result of hepatofugal blood flow caused by portal hypertension from liver cirrhosis.

Systemic and superficial skin infections, including bacterial and fungal infections, represent another health problem found to be more prevalent in alcoholics.2,4 The higher incidence of infections is likely attributable to multiple factors, including alcohol associated nutritional deficiencies in combination with immunodeficiency. Most notably, zinc and vitamin C deficiencies lead to poor wound healing, weakened mucosal barriers, and altered immune defenses with increased risk for infections. Group A streptococci, Corynebacterium, and Staphylococcus aureus are common bacterial culprits,2 as are fungal infections with various tinea and Candida species.2,5 Malabsorption associated with alcoholism is another mode by which alcohol can produce cutaneous abnormalities. Angular stomatitis, glossitis, perifollicular hemorrhages, pellagra, petchia, and ecchymosis are just a few such cutaneous manifestations.

**Skin Changes Directly Caused by Alcohol**

Porphyria Cutanea Tarda (PCT) is a metabolic disorder with cutaneous manifestations resulting from an aberration in hepatic heme biosynthesis. Whether acquired or inherited, PCT results from a deficiency in one of the hepatic enzymes involved in porphyrin metabolism, specifically uroporphyrinogen decarboxylase.2,3,6 The resultant upstream accumulation of photoreactive porphyrin precursors renders the skin extremely...
photosensitive. Alcohol is a potent inducer of the hepatic enzymes and the heme metabolic pathway, leading to an accumulation of photoreactive porphyrin compounds proximal to the enzymatic defect and, thus, precipitating PCT flare-ups. The cutaneous characteristics of an acute PCT attack include skin blistering and erosions on sun exposed areas that resolve leaving residual scarring and milia.

Alcohol impairs the vasomotor center of the brain, inducing peripheral vasodilatation. Hence, it has been suggested that this resultant cutaneous vasodilatation may exacerbate rosacea, contributing to the hallmark redness and flushing. Alcohol can also promote facial erythema in people without rosacea through a genetic deficiency involving an alcohol metabolism enzyme. This phenomenon is most commonly recognized in Asians, as studies have shown that 50% lack the ability to make aldehyde dehydrogenase, leading to an accumulation of acetaldehyde after alcohol consumption.

**The Role of Alcohol in the Pathogenesis of Psoriasis**

Psoriasis is a common chronic inflammatory autoimmune condition, affecting approximately 2% of the population in North America. It is characterized by epidermal hyperproliferation and a multifactorial etiology. A complex interplay between genetics and extrinsic factors, including the environment, trauma, infection, and social behaviors appear to be influential on the origin and clinical course of the disease. Extensive evidence demonstrates a link between excessive alcohol consumption and psoriasis. The amount of alcohol consumed and the type of alcoholic beverage have both been shown to confer the most risk for development and/or exacerbation of plaque psoriasis. A recent prospective study following 82,869 women for 14 years showed that consumption of more than 2.3 alcoholic beverages per week was a significant risk factor for new onset psoriasis. Furthermore, the same study found that consuming non-light beer appears to be an independent risk factor for developing psoriasis in females. Similarly, in males, excess alcohol consumption (at levels higher than 100g/day) appears to be a risk factor for the development and increased activity of psoriasis. Moreover, the misuse of alcohol in patients with psoriasis has been shown to be associated with decreased response to treatment. Interestingly, the cutaneous distribution of psoriasis in heavy drinkers tends to be predominantly acral, involving the dorsum of the hands and digits, resembling that seen in immunocompromised patients, such as those with human immunodeficiency virus (HIV) infection. This distribution highlights the potential role of alcohol induced immunosuppression in the development of psoriasis.

The exact molecular mechanisms by which alcohol triggers or exacerbates psoriasis are yet to be fully elucidated. One theory is that alcohol misuse may induce immune dysfunction with resultant relative immunosuppression. Alcohol may also enhance the production of inflammatory cytokines and cell cycle activators, such as cyclin D1 and Keratinocyte Growth Factor, which could lead to epidermal hyperproliferation. Additionally, increased susceptibility to superficial infections commonly observed in alcoholics, such as those caused by *Streptococcus* and trauma, has also been postulated to have implications in the development of psoriasis.

Although the environment and genetics may not be amenable to prevention or alteration, social behaviors such as alcohol consumption can be modified with appropriate counseling and pharmacological interventions, and therefore, appears to be a promising adjunct to the medical therapy of psoriasis.

**Conclusion**

An overwhelming amount of evidence suggests a significant link between alcohol and psoriasis - a multifactorial autoimmune disorder. Not only may alcohol contribute, in the presence of appropriate genetic makeup, to the development of psoriasis, it also results in more extensive and treatment resistant disease. Ascertaining carefully the presence of this risk factor in all patients suffering from psoriasis and providing appropriate counseling and education may help the clinician to minimize the risks of disease exacerbation and achieve better therapeutic outcomes.

**References**

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- PASItraining.com
- SkinCareGuide.ca
- SkinPharmacies.ca
- SkinTherapyLetter.ca
- SkinTherapyLetter.com

Social networking sites for patients and health care professionals:

- GenitalWartsPatients.com
- PsoriasisPatients.com
### Update on Drugs

<table>
<thead>
<tr>
<th>Name/Company</th>
<th>Approval Dates/Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gabapentin tablets</strong>&lt;br&gt;GRALISE™ (DM-1796)  &lt;br&gt;Depomed, Inc.  &lt;br&gt;Abbott Products, Inc.</td>
<td>The US FDA approved gabapentin extended release tablets in January 2011 for the once-daily treatment of post-herpetic neuralgia (PHN), the pain following herpes zoster infection (shingles). Treatment is to be titrated over a 2-week period to an 1800 mg once-daily dose. The tablet swells in gastric fluid and gradually releases gabapentin. This formulation is not interchangeable with other gabapentin products because of differing pharmacokinetic profiles that affect the frequency of administration. Furthermore, the FDA has granted GRALISE™ Orphan Drug status.</td>
</tr>
</tbody>
</table>

| **Oxychlorine compound**<br>Epicyn™ HydroGel  <br>Oculus Innovative Sciences, Inc. | The US FDA approved a new skin care gel in February 2011 for the management of burning, itching, and pain associated with various types of dermatoses, including atopic dermatitis and radiation dermatitis. The product is approved as a medical device and granted 510(k) clearance for marketing. This prescription product is intended for use under the supervision of a healthcare professional. In addition, the gel relieves the pain of first- and second-degree burns and maintains a moist wound and skin environment to help treat dry and waxy skin. The hydrogel is a shelf-stable hypochlorous acid formulation based on the proprietary Microcyn® technology platform. |

| **Collagenase clostridium histolyticum**<br>XIAPEX®  <br>Pfizer Inc.  <br>Auxilium Pharmaceuticals, Inc. | The European Commission (European Medicines Agency) approved this novel injectable treatment in February 2011 for Dupuytren’s contracture in adults with a palpable cord. It was US FDA approved in 2010 under the trade name of XIAXEL®. |

### Drug News

A recently published study* in the *New England Journal of Medicine* reported that the human papilloma virus (HPV) quadrivalent (types 6, 11, 16 and 18) recombinant vaccine (Gardasil®) is as effective at preventing HPV infection and genital warts in men as it is in women. The study enrolled 4065 healthy boys and men (aged 16 to 26 years) from 18 countries in a randomized, placebo-controlled, double-blind trial and found that HPV vaccination was 90% effective in protecting against HPV and genital warts. Although the reduction in precancerous lesions may reduce the rates of penile and anal cancers in males, their incidences are significantly lower than cervical cancer. This investigation does not address the cancer risk in the specific age-gender group studied.

Both of the HPV vaccines (Cervarix™ and Gardasil®) are recommended for females aged 9 through 26 for the prevention of cervical cancers caused by HPV types 16 and 18 (high risk types), which cause most cervical cancers. These vaccines are now on the US Centers for Disease Control and Prevention’s (CDC) routine childhood vaccination schedule for girls starting at age 9. Only one of the vaccines (Gardasil®) also protects against HPV types 6 and 11, which cause most genital warts in females and males. The quadrivalent vaccine is recommended for optional use in boys and men. Gardasil® was approved for use in males 9 through 26 years of age in 2009 by the US FDA, however, the CDC’s advisory panel voted against routine use of the vaccine in boys and men. More information on the CDC’s recommendations is available at: [http://www.cdc.gov/vaccines/vpd-vac/hpv/vac-faqs.htm](http://www.cdc.gov/vaccines/vpd-vac/hpv/vac-faqs.htm)